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THE GUMMOSIS OF SUGAR CANE

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This disease was first found in Porto Rico in February, 1920 by Matz (**13, 14, 15**) who reported it from the neighborhood of Trujillo Alto, Río Piedras, Guaynabo, Las Piedras and Morovis. By January 1921 it had been reported from a much larger area including Caguas, Cidra, Cayey, Barros, Corozal, Aibonito and Barranquitas. In 1922 the disease was reported from Adjuntas, Isabela, Peñuelas, Guayanilla, Yauco, Humacao, Fajardo, Río Grande, Carolina and well to the west of Bayamón. Most of these points are in the eastern half of the Island. Matz (**1**) reports finding the disease on the oldest varieties grown on the island, such as Otaheiti, Rayada, Cristalina and Cavengerie or Colorado. The experiments and reports of Mr. Matz will be referred to later.

Mr. Matz left the Insular Experiment Station in 1922 and the writer took charge of the plant pathology laboratory in July 1923 which was too late to get records for that year. No severe attacks of the disease came to our attention until the spring of 1925 when a serious outbreak on the property of Central Columbia, Maunabo, was reported to Commissioner Chardon and the writer. Examination of the records of this Central showed a lowering of purities and other troubles over a period of about eight years which were probably due to this disease. The Cristalina which was grown most extensively on the properties of Central Columbia and vicinity was found to be severely infected. We recommended the substitution of BH-10 (12) and SC-12 (4) for the Cristalina which has resulted in the elimination of the troubles and an increase in sugar production. The Company very generously set aside one and one-half acres of good land for our use in testing varieties and have given us every possible assistance in this work. The planting and cultivation of these experimental plots was under the supervision of Mr. Francisco Ortiz.

GEOGRAPHICAL DISTRIBUTION

This disease has been definitely reported from New South Wales, Queensland, Fiji Island, Mauritius, Java, Borneo, New Guinea, Brazil, Colombia and St. Kitts of the British West Indies. A careful study of other cane growing regions of the world may show a much wider distribution. Some writers have expressed the opinion that the disease was in Cuba but these opinions appear to be without foundation.

HISTORY [1]

It appears that this disease was very severe in the province of Bahia, Brazil, as early as 1863 but the first published report was by Dränert (**9, 10**) in 1869. The disease, like many others, having been ascribed to insects, Dränert made some studies on this line with negative results. However, he did find several minute organisms in the diseased cane which he supposed to be the cause of the disease. Following Hallier's idea of polymorphism, he supposed all these organisms to be stages of a single species. His description of the symptoms and the yellow gum leaves very little doubt that this is the first authentic record of this disease.

The disease was next reported from Australia in 1893 by Cobb (**3, 4**) who was of the opinion that it had been in the country since 1876. The discovery of the disease in these two widely separated parts of the world is of special interest. He described the organism which he discovered under the name of *Bacillus vascularum*. He conducted many successful inoculation experiments but did not use pure cultures. However, there is no doubt as to the authenticity of his records and the disease is frequently referred to as "Cobb's disease of Sugar Cane".

In 1894 the disease was again reported from Brazil (**17**) but was much less severe than in 1869 which was supposed to be due to the growing of more resistant varieties. This attack was on Otaheiti (known in Brazil as Cayanna) which we now know to be one of the most susceptible varieties. We have no very definite information as to the resistant varieties grown at that time.

In this same year the disease was reported from Mauritius by Bonâme (**1**) as common on La Canne Bambou.

Extensive studies were made in Australia during the next few years by Cobb (**6**), Tryon, (**22**) and R. Greig Smith (**18, 19**) which added to our knowledge of the subject.

[1] The facts concerning the history of this disease are drawn largely from Smith's Bacteria in Relation to Plant Diseases (**21**).

Dr. Erwin F. Smith published a short paper (**20**) in 1904 but nothing more appears to have been done until this same author published the results of a very comprehensive study (**21**) of the disease in 1914.

This was followed by studies in the Dutch Indies by Groenewege (**11, 12**) and Wilbrink (**23**) from 1915 to 1920.

The next studies were made by Matz (**13, 14, 15**) in Porto Rico in 1920 and 1921.

The disease was found in St. Kitts of the British West Indies in 1925 (**26**).

The last publication which has come to the attention of the writer was by Chardon (**2**) who found the disease in Colombia, South America in 1926.

ORIGIN AND SPREAD OF THE DISEASE

It is frequently very difficult to trace a disease to its origin. The sugar cane like many other of our important crop plants is a product of Asia. In its migrations to other parts of the world it has no doubt carried many of its diseases and has probably contracted other diseases from related plants in the course of its wanderings. Although we do not know where the disease originated, it is interesting to note that the first three reports of this disease were from three distant parts of the world, Brazil, Australia and Mauritius. It is improbable that the disease originated independently in these three distant places. North (**16**) is of the opinion that the disease was brought to Australia from Brazil or Mauritius. The geographical study of this disease is further complicated by its behavior in the field. The first report from Brazil in 1869 indicated that the disease had been in the country at least six years previous to the report. Cobb's first report from Australia in 1893 indicates that it had probably been in the country for seventeen years previous to that date. Cobb also states that the symptoms were much more pronounced on plant than on rattoon cane. North states that the disease sometimes disappears from localities where it had been very severe.

Matz comments on the sudden appearance of the disease in Porto Rico as follows: "From the sudden appearance of gumming disease here in the fields, where no new seed introductions were made for some time and in varieties which have been grown here for many years, no definite opinion could be formed as to its future possible spread, but now it is certain that it is spreading rapidly and that it is becoming a general epidemic." The wide distribution of the

disease in Porto Rico justified this statement by Matz but the very few reports during the next two years indicated that if the disease was present that it was in such small quantity as not to attract the attention of either the growers or the mill men. During this time many suspicious pieces of cane were sent to the writer for examination but only a very few proved to be infected with the gum disease organism. In fact the only locality in which the disease persisted in quantity was the vicinity of Fajardo and even there it was greatly reduced.

In the spring of 1925, the writer and Hon. C. E. Chardon, Commissioner of Agriculture, were called to Central Columbia at Maunabo where we found a severe outbreak of gummosis on Cristalina and Rayada, the two most important varities grown in that immediate vicinity. A study of the mill records showed more or less lowering of purities over a period of about eight years which may have been due to this disease. Acting upon recommendations the owners of the properties on which the disease was present we started a campaign for the elimination of the Cristalina and Rayada and the planting of BH-10 (12 and SC-12) (4). In 1928 this Central reported the largest production of sugar in its history from a smaller acreage.

These results from the two Colonias from which this Central drew the largest amount of its cane may be summarized as follows:

	1924-25	1925-26	1926-27	1927-28
Percentage of infection	37.8 36.3	2.0 3.5	12.4 16.	8
Rainfall inches	85.19	62.00	71.35	77.93
Total acreage	1056.63	825.71	750.00	769.56
Cane tonnage	30500.00	18664 00	25519.30	36188.99
Bags Sugar	48544.00	56544.00	41589.00	75894.00

Reports over 1925 to 1927 indicated small infections of the disease from Santa Isabel on the west to Fajardo on the east, but no severe outbreaks were reported. In 1928 the disease was reported from Fajardo and Santa Isabel and the symptoms were found on two seedlings in a field test west of Ponce. A study of the field test (page 167) Central Columbia showed a decided decrease in the amount of infection the second year as compared with the first year. This is in harmony with reports from other parts of the world. The writer has no explanation for the sudden appearance of the disease in Porto Rico or for its fluctuations in Porto Rico and other places.

Some doubt exists as to whether the disease is the same in the

various countries from which it has been reported, especially the form appearing in Java. The form appearing in Java does not produce gum as in the case of other countries. The writer agrees with Wilbrink that a comparative study of the two forms of the disease must be made before this point can be determined.

Cultures of *B. vascularum* were sent to Dr. M. C. Goldsworthy of the University of California for comparison with cultures from Australia. He replied as follows:

BERKELEY, CALIFORNIA, *June 27, 1927.*

DR. MEL. T. COOK,
Insular Experiment Station,
Río Piedras, P. R.

DEAR DR. COOK:

I have now your *Vascularum* Cultures. They arrived about a month ago, to be exact on the 24th of May. The cultures were in excellent shape, excepting Nos. 5 and 6. I have tried everything to invigorate them but to no avail. Your *vascularum* types are different than those that we have received from Australia. That is they behave differently on media. So far I have had no opportunity of comparing the cultures by the serological method.

In am expecting cultures of *B. stewarti* and *Ps. campestris* from the type culture collection at Washington to compare with your types and with other yellow organisms.

Hoping that you will not become impatient for results and thanking you for your kind assistance in getting these valuable *vascularum* cultures I remain,

Very truly,

(signed) M. C. GOLDSWORTHY,
Research Assistant.

Unfortunately were not able to follow this comparison of cultures which leaves us in doubt as to whether the Porto Rico organism is exactly the same as the one found in Australia.

SYMPTOMS.

The first reasonably authentic description of the disease was given by Cobb and is as follows:

"When a cane crop is gummed it presents a variety of symptoms that vary according to the severity of the disease. When only slightly gummed the crop appears to be in a fair condition. Here,

and there, however will be seen stools containing one or more stalks with dead tops. The base of the arrow (terminal shoot or flower-stalk) in such cases will be found to be rotten, and usually one or more cavities of considerable size are to be seen near the top of the stalk, filled or partially filled, with offensive matter. At first one is inclined to attribute these cavities to the inroads of grubs or borers. This idea is soon seen to be mistaken, for there is no entrance or outlet to the cavity, nor are there any traces of excrement, both which facts prove conclusively that the cause lies in some other direction. The tissue about these cavities is generally brown, black, or dark red in color, and reeking with a slimy offensive substance which varies from nearly colorless, through yellow, to brown. Plants whose tops have died from gumming often shoot from buds half way down, but this symptom is not peculiar to gumming. It occurs also in plants nipped by frost or by borers''.

''If a stock which has died at the top in the manner described be cut into pieces with a very sharp knife, in such a manner as to leave the cut surface quite smooth, a honey-colored gummy matter will, in a few minutes, be seen to ooze slowly out and form in droplets on the end of the cut fibers. This gum is sometimes nearly transparent, sometimes rather opaque, and varies also in color from nearly colorless into various tints of yellow according to the stage reached by the disease. This gummy matter is usually more abundant near the top of the stalk than the near the bottom, or at least oozes out more freely. In the course of an hour or thereabouts these droplets of gum become so large as to run together and form large drops, and if two or three dozen cuttings from badly diseased stalks be laid in a closely covered box over night one may in the morning collect from their ends a teaspoonful of yellow mucilaginous gum.''

''The gum which thus oozes out in such quantities, if allowed to do so, at last becomes dry, owing to the evaporation of its water. In this state it appears as bright yellow stains on the end of the cutting. Sometimes the gum oozes out in a state so nearly dry that it hardens as it issues from the fibers into a yellow, coiled-up, hair-like body, and inasmuch as each fiber gives rise to one such body, all the fibers together originate a yellow mossy appearance on the end of the cutting.''

''If another stalk, which is apparently sound, be taken from a stool in which one or more stalks have been already blighted by gumming, as above described, such stock will be found to exhibit the same symptoms, except that the cavities and rottenness at the base

of the arrow are wanting. Moreover, if other stalks be removed from shoots of cane standing nearby in the same field, but which as yet, show no outward symptoms of this disease, the chances are that some of them will also be found to be more or less gummed."

"In some cases the amount of gum is so small as to ooze out in but a trifling quantity, to be detected only with a magnifying glass. Finally, the quantity of gum may be so small as not to ooze out at all; in such cases a good microscope is necessary to demonstrate that the gum is present."

"After the cane is cut and ready for the mill, gummed stalks can be recognized not only by the yellow dried-up gum that exists on the cut ends, but also by their color. Gummed stalks generally have an over-ripe appearance. Green and yellow canes, when badly gummed become yellowish or orange, or even somewhat purple in color. The ribbon canes show similar alterations in their yellow stripes while their purple or black stripes tend to take on a reddish cast. The waxy bloom, usually to be seen on perfectly sound cane, has disappeared on badly gummed cane. All symptoms of gumming seem to be more pronounced when they occur in plant cane than when they occur in ratoons. An explanation of this fact will be offered later on."

"In the sugar mill the juice of gummed cane may be recognized by the greater amount of lime required for its clarification, and there seems to be reason to suppose that the crystalization of the jellies is slower and less perfect when they are derived from healthy cane. The sieves used to separate the fiber from the juice are apt to clog up when gummed cane is being crushed. They are easily cleaned with soda."

"The foregoing symptoms are those connected with cane so attacked as to produce a marketable crop. In some cases, however, the gumming prevents the growth of the sets. The plants may reach a height of a foot or two feet, but they then die back and shoot again from the base, or from buds half-way down the stalk. In such a case the loss is total or nearly so. The gum presents the same features in this case as in milder ones."

"A microscopic examination of a thin slice across a gummed cane shows at once that the disease is not general, but local. The gum, except in certain cases, is confined to the fibers; in fact, to the sap-vessels, these latter being plugged up with gum. A cross-section of a healthy fiber shows the sap-vessels as empty spaces, the sap having flowed out in the process of cutting the section; a cross-

section of a gummed cane on the other hand shows the sap-vessels to be filled with yellow granular matter, in other words, gum. This confinement of the gum to the sap-vessels is one of the most striking microscopic features of gummed cane. In advanced cases, and in the more tender tissues at the top of the cane the gum is not so local in its distribution; it may, under such circumstances, be found outside the fiber."

"Lenses of high power show the gum to be swarming with microbes of the form known as bacilli. When the gum is fresh and yellow in appearance, the microbes are all of one kind whose features are well shown in the illustrations. This microbe appears to be one not hitherto described, and I propose to call it *Bacillus vascularum*, in consequence of its occurrence in the vessels of the sugar cane. Each microbe has about it a small amount of gummy matter, which is a product of its growth. The gum described above as issuing from the sap-vessels of the cane has, therefore, two component parts, namely microbes and viscous gummy matter. This gummy matter appears to be a new substance, and to it I have applied the name vasculin."

"Vasculin, the substance formed by the growth of the *Bacillus vascularum* as it occurs in sugar cane, is a yellowish, non crystallizable, viscid substance, having an almost imperceptible acid reaction. The taste is that of a slightly soured solution of gum arabic. A short time after oozing out from the ends of cut cane, by solution it converts ten times its weight of water into a fluid of the consistency of mucilage as used for adhesion purposes. Though soluble in water it is insoluble in alcohol. The addition of absolute alcohol to the raw substance converts it into a hard mass, but this is only owing to the abstraction of water; on placing the hardened mass in water it soon resumes its former consistancy and appearance. Vasculin is not coagulated by alcohol".

The description of the disease in Pernambuco, Brazil, in 1894 is as follows:

Gum—"The formation of a bright yellow, gummy substance, exuding apparently from the ends of the fibres on cutting the cane across. Sometimes this yellow gum, which turned orange colour on drying, appeared only after half an hour or more, at other times it appeared immediately on cutting the cane, and in such quantities as to drip from the cut ends, and in very bad times is said to have frequently checked the strainers of the raw-juice tank."

Premature death—"Indicated by the early drying up of the leaf

tips; by the successive joints becoming shorter and less in diameter as the cane grows, giving it the appearance of the joints having been telescoped one into another; by some internal parts turning red, denoting fermentation, which may have set in before or after the cane had succumbed, and by the death of the eyes."

"Any or all of these symptoms may have occurred together, and some canes, which to all outward appearance were perfectly healthy and well developed, contained a quantity of the yellow gum, together with a normal percentage of sugar".

"The canes which did not die before maturity gave no ratoons, and the juice was most difficult to work, refusing to crystallize in the ordinary way, sometimes in any way. The greatest difficulty was encountered in the 'old process' factories, probably on account of the great heat accompanying open evaporation. The Usines, with their lower temperatures, could work better, but they also at times got the 'devil in the house', especially if they did not take care to use but little lime in the defecation. The lime was frequently reduced to 10 grammes per hectolitre, and in this way the juice worked best".

Tryon described the disease as follows:

"When the presence of the disease has been determined by the use of sets derived from a badly 'gummed' crop, some of them will fail after having emitted attenuated shoots, that fail to reach the surface. Others will give rise to slender and weakly plants, which apparently struggle to survive, whilst a few will develop foliage with the individual leaves narrower and more irregular in size than if they were healthy, with the control ones yellow green and more or less crinkled and contorted, with at times, the central leaves interlaced in a tangled mass. A few of these leaves again, specially the inner ones, may exhibit bright rust-red streaks in their tissue, and this may constitute a conspicuous feature. If any such plant is cut longitudinally through the point of growth, it will be generally observed that the central shoot beyond where it joints the cane proper, instead of arising in a straight, erect manner, is more or less bent and contorted, its upward tendency having been apparently hindered. Moreover, the young cane itself, immediately below the shoot, will present one or more cavities, each containing a semi-fluid tenacious pale brown substance."

"Should the 'sets' be the progeny of a crop of cane affected but slightly by the disease, nothing very abnormal may be noticed until the cane proper has commenced to form. Then, although the

bulk of the plants will appear well-grown and perfectly healthy, others, though at first fully developed, will evince the presence of the affection in varying degree. The first symptom in a plant of its occurrence will then probably be afforded by a thin, pale longitudinal stripe arising in one of the outer leaves, in which the green colouration being discharged may gradually be giving place to brown. If this plant is divided by a longitudinal cut through its growing apex, nothing abnormal may yet be recognized. A plant in which the disease has made further progress will exhibit brown stripes of dead tissue one side of the central nerve, or along the margins of the outermost leaves, whilst on two or three of the inner ones will have appeared elongated rust-red streaks. If this plant be cut as before, very marked changes will be noticed in the tissue immediately below the growing apex. These are afforded by the presence of several cavities having ill-defined walls partly filled with an odorous yellowish substance of the consistence of pus, as well as of spots where the external tissue is becoming soft and brown to mark the site where subsequent ones may arise. These cavities occur in the intervals between nodes, whilst the denser tissue of which these latter are composed may exhibit—in small number—specks, or thread-like lines of a red colour. When still further advanced we may have a strong, healthy looking plant, with a stem measuring some 2 feet 6 inches in height from the ground to where the green foliage commences. In this several of the outer leaves, and the central ones as well, have longitudinal brown stripes or bands of dead tissue extending for the greater part of their length; and, as will be seen by a longitudinal cut, the joints or internodes immediately below the apex, to a distance of from 1 to 2 inches, are quite hollowed out, and there is considerable discolouration and softening of the tissue generally and incipient decay, both in them and in the nodes also. In such a plant the central shoot, now almost dead, may readily be pulled out."

"The disease may appear at any period in the growth of the cane, and even when it is fourteen or more months old; but these late manifestations probably generally arise when the malady is of spontaneous origin. In the first instance the central leaves are quite normal in appearance, and perfectly green and turgid, but the lateral leaves are marked by broad brown longitudial bands of dead tissue the lowermost having evidently prematurely died. The eyes on that part of the stem from which the leaves have naturally fallen have shot out, and the slender shoots thus formed are already dead;

the uppermost eyes, however, are still alive, and have not as yet sprouted."

"As an illustration of a still further advance in the progress of the malady, a plant may exhibit the following symptoms: The central shoots and leaves are already quite dead, though still flaccid, and readily yield to a slight pull. There occurs also in those, on either side of it, that are still partly green, a brown band of dead tissue, proceeding along each margin; this band widens in the case of the outer leaves, whilst the outermost of all are all involved in it, being quite dead. The cane itself, instead of exhibiting that yellowish colour indicative of the fact that it is already ripe, is of a dull bright-green colour above; nearer the ground it is clouded, with the same hue, but at the extreme base evinces little unusual in this respect. All the buds except those situated lowest on the cane have sprouted, some having given rise to peculiar elongated slender shoots; beneath the leaf-sheath the buds are already dead or apparently dying. * * * * * On cutting any of these canes across, small bright droplets of a canary-yellow thick adhesive substance will arise from the pores distributed over the surface of the section, and in many instances will run together and coalesce. When the disease is advanced to the stage above described, some of the buds within an inch or so of the decaying summit of the shoot may shoot out and develop a tuft of narrow leaves; but no further growth takes place in the cane itself, though the latter may be some time before it actually dies."

"All the canes that arise from a single stool may not be affected simultaneously and to an equal extent. As an instance of this, the following occurrences were remarked in the case of an affected plant: All the canes had been checked by cold when they had experienced from six to seven months' growth, otherwise the plant was remarkably robust. Three of them had the central shoot, and several of the lateral ones already dead. A fourth had the leaves as well as the central shoot quite green, and all of these canes exuded droplets of gummy substance when their stems were cut across. A fifth cane—the stoutest of all—was entirely wanting in the gummy exudate, as was seen when after lopping off its top and allowing it to remain still connected with the ground, none of this gum was observable even after the lapse of some hours. In addition to these five canes, there were several suckers, some of which had formed cane, and were almost as high as the stalks that surrounded them. These, however, even when arising alongside

gummy canes, were themselves apparently quite free from disease, as no 'gum' emanated from their cut ends."

Smith described the disease as follows:

"The most conspicuous signs of this disease are dwarfing, striping of the leaves, dying of the tops, decay of the heart, (terminal bud), and the appearance of a yellow slime or gum in the bundles of the stem and leaves. Many of the bundles are also stained red. Microscopic examination shows that this gum contains millions of bacteria. Cobb and Bonâme agree that there is also a reduction of the sugar-content".

"The disease is primarily one of the vascular system, but in advanced stages the parenchyma is attacked, especially the soft tissues just below the terminal bud, and cavities are formed which are filled with the yellow bacterial slime. Sometimes these cavities contain as much as a teaspoonful of the slime. In the later stages of the disease, the interior of the leaf-sheaths is rusty brown and covered with the sticky bacterial slime, which is also sometimes seen oozing from other portions of the leaf. This slime oozes from the stomata. In very bad cases the leaf-sheaths above the terminal bud are completely stuck together, so that the growing shoot can not elongate naturally, but is forced to bend on itself repeatedly and push out sidewise through the sheaths. The gumming together and pressure of the outer leaves around the terminal bud result in the doubling, twisting, and bulging of the main axis and eventually the stopping of the terminal growth. In the most pronounced cases the terminal shoot enveloped in its wrappings has a club-shaped appearance. In such cases there is sometimes a development of lateral shoots and of aerial roots".

Matz describes the disease as it appeared in Porto Rico as follows:

"The principal symptom of the disease is the yellow gummy exudation from the cut ends of the affected cane, and it is so striking that few can fail to become aware of its presence. The exudate varies somewhat in color and abundance. At times it is grayish yellow and somewhat watery but more often it is lemon yellow and thickly gummy. In almost every case, and especially where the disease was present in any marked quantity of the harvested cane, the mill men recognized the disease by this symptom before their attention was called to it. This helped to ascertain the distribution of the disease."

"At first it was thought that gum-disease cane could be recog-

nized only by the yellow gummy exudation from the cut ends of the cane, but further observation showed that the disease can be located in growing canes, before they are cut, by a peculiar appearance in the leaves. The leaves, and mostly the younger and innermost not fully unrolled ones, show, in the early stages of the gum disease, pale green to almost pure white patches and longitudinal bands or streaks. These light-colored areas become often sprinkled with dark-red small spots or narrow and short streaks. Such leaf symptoms can be found in young shoots or in older ones in the not quite unfolded basal parts of their inner leaves. In the outer maturer leaves long dark brown streaks may be found. In older cane and where the disease is more advanced the inner leaves possess long, sometimes lighter and sometimes darker gray, dead, stripes usually about 1 centimeter in width. These stripes are usually found towards the middle of the leaf blade. This feature distinguishes this symptom from the ordinary drying of leaves which occurs in cane either because of white-grub injury, borers or drought. In the latter cases the edges of the leaf commence to dry first. In gum-diseased cane the dry stripes are usually in the interior of the leaf, while the edges may remain green for a long time. This phenomenon is due primarily to the partial infection of some of the fibers; naturally only the cells surrounding the infected fibers die first and result in the dead-stripe appearance in the leaf. Usually the tops of gum-diseased cane showing the dry stripes will not be as widely unfolded as in healthy cane, the dead longitudinal areas or stripes in the leaves preventing the straightening out of the leaf blades, therefore the tops in gummy cane usually stand up erect and are more or less unfolded. In the latter stages of the disease an odorless decay sets in the tissues of the growing points of the cane. At this stage the outward symptoms bear resemblance to the top-rot condition of cane caused by borers and Plasmodiophora disease. In the last cases the cause of top rot is due entirely to the interference with the normal functioning of the fibro-vascular system. However, whether it is a bacterium as in gum disease or a Phasmodiophora as in dry top rot which fills up the water-conducting vessels, or whether it is a mechanical cut made by an insect, thus breaking the connection between the roots and top of growing point where the new leaves issue from is the same. In gumming disease, in addition to a clogging of the fibers, there is a direct decay of the tender tissues of the top caused by this bacterium as is evidenced by the red coloration of the tissues between the fibers.''

"Another phenomenon found with gum disease is the red coloration of some of the fibers themselves in severely affected cane. This is not a primary symptom of gum disease, but it indicates that the phloem in some fibers, or in these which show the red color, have died. Canes which show exudation of gum from a majority of their fibers do not in many cases have a single red-fiber. In many instances a severe stunting of the stalks and the presence of grayish longitudinal depressions along the internodes was quite common. In gumming top rot there may be present masses of gum between the leaf sheaths and the stalk."

"In summarizing the symptoms of gum disease as it occurs in Porto Rico we must distinguish between the primary and secondary symptoms. The primary symptoms, or those which are always associated with gum disease, are the yellow exuding from the fibers of cut canes, the light areas sprinkled with dark-red little streaks in the younger portion of leaves, brown long streaks and light to dark gray, more or less wide dead stripes in the older leaves, and top rot. Red fibers, and even stunting of cane may or may not occur in gum-diseased cane, depending on the severity of the infection. While the gum-flow symptom is the easier to detect after the cane is cut, the leaf symptoms are more important, because by these it is possible to detect the disease in the field before the cane is cut, and it should be taken advantage of in controlling the disease. Wherever possible, diseased stools should be cut after the healthy stools have been harvested. This is hardly possible in severely infected fields, but it should be borne in mind that infected machetes can introduce the disease into healthy cane."

Cottrell-Dormer describes the disease as it occurs in Queensland, Australia, as follows: "The symptoms of gumming disease in young cane vary greatly, but the most characteristic sign is the presence of large, irregular, longitudinal white patches on the young leaves. These are seen shortly, after the leaves first appear from below the soil if searched for, and are readily observed when the plant attains an age of two or three weeks. Similar signs sometimes occur on the young cane, but any doubt on the true nature of these marks may at once be settled if the set is dug out, cleaned and cut transversely into three pieces; these pieces should be placed into a closed "billy can" for a few minutes; if gumming disease is present the yellow drops of gum so characteristic of the disease will be seen to have exuded from the cut ends of the pieces. Now these plants which show these white patches on the leaves are only those

which were planted from very badly diseased canes, so that for every one of these evidently diseased plants will be a great many which were planted from slightly diseased stems, and probably will not show the disease until the cane is a great deal more mature".

The same author also describes the leaf symptoms as follows:

Symptoms.—"The leaf symptoms enable a quick and reliable diagnosis, but they do not appear at certain seasons. The "gum streak" bears a superficial resemblance to streaks due to other causes. Typically, it is a yellow streak from ⅛ inch to ¼ inch in width, of varying length, running along the veins to the leaf margin. It is almost invariably dotted with dull red, and, as it matures, becomes converted into dead tissue at that portion of the leaf where the streak originated. The streak usually begins at the margin, and works down towards the leaf sheath, but sometimes originates further down the leaf, and then elongates in both directions. The dead tissue at the point of origin spreads, and is delineated from the living by a dark red or brown margin. The gum streak is best observed between a fortnight and eight weeks after good rain has fallen; for during the warm rainy season the growing conditions of the cane are often such that the streaks do not appear. Again, after prolonged drought, the older leaves having withered, the young leaves formed no longer develop streaks. At this time the cane which is badly infected will wilt, and perhaps die. The critical symptom—the oozing of gum from the vascular bundles—should then be sought. The stalk is always pulled out, to avoid knife infection, and a portion freshly cut ends is placed in an enclosed space (a billy can) to prevent premature drying. The globules of gum which are "sweated" out constitute the critical symptoms of gumming. A stalk will not "sweat" gum unless it shows reddening of some of the fibres at the nodes, in a longitudinal section of the stem. A stool dying from gumming will always "sweat" gum; but in other cases if gum does not ooze the stool may be recently or lightly infected, or the growing conditions may be too moist for the gum to be evident."

The writer of this paper has made a careful study of the symptoms on field cane, in experimental plots and in the green house and has compared notes with the preceding descriptions. The results of these comparisons are as follows:

1. Poor germination which varies with the severity of the infection. Poor germination is also due to other causes.

2. "The doubling, twisting and bulging of the main axis" referred to by Smith is rare and may be due to other causes.

3. The white patches on the leaves of young plants (Fig. 2–6 & 17) described by E. F. Smith and by Cottrell-Dorner occur in less than one per cent of canes grown from infected seed. I doubt if it ever occurs except on canes from severely infected seed. The writer sectioned some of these white areas and found the cells filled with bacteria. However, it is well known that chlorosis may be due to other causes.

4. The bent and contorted tips described by Cobb are rare.

5. The most reliable external symptom is the leaf streaks (Figs. 7–10) referred to by Matz and Cottrell-Dorner. However, this symptom is sometimes present on the POJ canes, and occasionally on Uba and some other canes, although it is impossible to find any trace of gumming in the cut surfaces. The writer has found the organism in these streaks on the leaves of infected canes.

6. The dying of the tops occurs in severe cases and may be due to any one of many other causes. The gumming of the top leaves occurs in cases of very severe infection.

7. Young canes die in the infected stools in numbers varying with susceptibility and severity of infection. The "eye spot" (*Helminthosporium sacchari,* Butler) and the dry top rot (*Plasmodiophora vascularum,* Matz) will produce the same symptoms.

8. The red fibro-vascular bundles which have been referred to in gum canes are so common in sugar cane that they must be considered of little or no importance as a diagnostic character. The writer has examined and made cultures from the discolored tissues in a large number of canes without finding any evidence of gum or bacteria.

9. The presence of the gum (Fig. 1) is the only sure sign of the disease. The statement by Cottrell-Dorner that "a stalk will not sweat gum unless it shows reddening of some of the fibres" is not true in Porto Rico.

The writer offers this description of the symptoms of the gum disease as he has found them in Porto Rico. The description is drawn from a study of the varieties referred to in the tables of this paper.

(1) The most reliable external symptom of sugar cane gummosis is the presence of leaf streaks (Figs. 7–10) which usually appear soon after the nodes are visible above ground. These streaks appear soon after the leaves are fully unrolled and usually start

at the margin and work inward along the fibro-vascular bundles but sometimes start in the interior of the leaf and work in both directions They are rarely more than ⅛ inch in diameter and are usually yellow or light green. A little later they develop reddish dots (Fig. 8) usually arranged irregularly in two lines. These dots may occasionally appear in the green tissue without being preceded by the yellow streak. This reddish color increases, becomes brownish and the tissues finally die. (Figs. 9–10). The dead brownish strips of tissue referred to by Matz are common but much less reliable than the yellowish streaks and reddish dots. This symptom may be present on POJ and occasionally on other canes which do not show the slightest trace of gumming.

(2) In older canes, broad stripes of dead tissues (Fig. 10) extending from the margins into the leaves may be sufficient cause for suspicion on susceptible varieties and in localities where the disease in known to occur. This stage usually follows the yellow streak, but sometimes appears in healthy canes.

(3) Diseased canes give a low germination and the young plants are occasionally twisted and doubled. A very few plants show broad white stripes and white areas. Brownish red dots frequently develop in these cholorotic plants. Plants of various ages and sizes die as a result of the disease and these plants are very soon attacked by the so-called rind disease fungus (*Melanconium sacchari* (Cke) Mass.)

(4) The dying of the tops is characteristic in the case of canes that are severely infected, but this symptom may be due to any one of many other causes. The development of shoots from lateral buds is common in severely infected canes but may be due to any one of many other causes. The formation of new shoots from top of diseased cane may be due to other causes as well as to gummosis.

(5) The formation of a honey yellow gum (Fig. 1) on the cut surfaces of infected canes is the only sure indication of the disease. In slightly infected canes it may develop as small drops on the cut ends of the fibro-vascular bundles but in severely infected canes the entire cut surface may be covered with a thick layer of gum. The variation in color of the gum, the fibro-vascular bundles and other tissues may be due to other causes.

(6) The leaf symptoms are more pronounced during wet than during dry weather. During periods of drought there may be no leaf symptoms on cane known to be infected, but these symptoms will be developed very rapidly following a heavy rain fall.

CAUSES

It is very generally recognized that "the disease is caused by *Bacterium vascularum* (Cobb), Greig Smith, which Dr. Erwin F. Smith describes as a 'honey yellow', one flagellate organism, which forms the yellow slime always present in the vessels of diseased plants. It is a short rod, occurring singly, in pairs, fours, or eights (end to end) and it often exists in practically pure culture in the fibro-vascular bundles of the diseased sugar cane."

The Organism

The causal organism can be found in great abundance in the gum which oozes from the cut surface of infected cane. In cases of severe infection a teaspoonful of gum be may scraped from the cut surface while in the case of mild infections the gum oozes out from one or more fibro-vascular bundles in small drops.

The gum has been described by Dr. Erwin F. Smith and others as "honey-yellow", but we found it varying from perfectly clear to milky white, to many shades of yellow, and occasionally orange, red and brown. Our inoculation experiments were not extensive but they indicate that the organism in all cases was pathogenic. Although Cobb describes the gum as yellow, he also states that "this gum is sometimes nearly transparent, sometimes rather opaque, and varies also in color from nearly colorless into various tints of yellow according to the stage reached by the disease". The writer is inclined to believe that the variations in color may be due to many causes such as acidity of medium, age, variety of host, etc. The red color appears to have been due in some cases to the influence of a very small fungus sometimes found growing in the tracheary tubes When the organism was separated from this fungus in culture, the bacterial growth resumed the white or yellowish color. The fungus appears to belong to the genus *Fusarium* or near related genus. It is white but produces a red color in the media. It grows in the tracheary tubes but is difficult to demostrate because it clings very closely to the wall.

Both Cobb and E. F. Smith state that the gum is the product of the organism and not of the desintegrating cells of the host plant. The writer agrees with this statement.

Several writers refer to the drying of the gum on the cut surfaces. This is also true but it is very soluble in water and easily removed by rain.

The culture made from the second cutting of our field experiments (1928) showed a much larger number of clear and milky colonies and a much smaller number of the yellow, red and brown colonies.

A red bacterium which was very common in the cultures was very distinct from the *B. vascularum*. It was not pathogenetic but appeared to live saprophytically in the fibro-vascular bundles.

The cultures involved the use of sixteen different media containing various mixtures of cane sugar, cane juice, glucose, peptone, $K_2P_2O_4$, $MgSO_4$, beef extract, oat meal and potato. The organism grew in both large and small colonies which were always semi-liquid in texture and raised above the surface of the medium.

The growth in the media varied to some extent with the severity of infections in the canes from which the cultures were made. The colors remained very constant except in the case of the red color to which we have referred and the yellow which sometimes became white when transferred to the beef extract medium. The organism retains its vitality in culture for more than twelve months but the growth becomes less vigorous with time. The writer also found the living organism in dead seed pieces two months after planting. These infected seed pieces had been planted in pots for experimental purposes but were so severely infected that they died without producing shoots. The gum in the seed pieces at time of planting was yellow but the gum in the dead pieces was very clear. The organism from these dead canes was capable of producing the disease.

The prevalence of other colors in the gum and in the tissues is so common that it is worthy of special attention. Smith (**21**) says: "In the majority of these red bundles bacteria were no longer to be seen. In place of them was a red formless mass. Red and yellow bundles were inter-mingled in the stems; however, often the same bundle would be both red and yellow, *i. e.*, variegated, the yellow parts being filled with bacteria. The same phenomenon has been observed in maize, inoculated with *Bacterium stewarti*, except that in the latter case the variegated bundles were yellow and brown. The red pigment was most pronounced in the nodes and immediately under them. This was observed in many canes. Without exception there was more pigment in the upper part of the internodes than in the central or basal portion, but by far the greater part was in the nodes, where often nearly all of the bundles

were as red as blood. Probably this localized pigmentation is due to greater aeration through leaf-traces centering in and immediately under the nodes. In the sugar cane, as in the sweet corn, the pigment does not appear in the first stages of the disease, and the writer is inclined to think that the reddening of the bundles is a later stage than the yellowing. Valeton makes the same observation respecting Sereh. Plates poured from bundles showing red ooze yielded only the yellow colonies of *Bacterium vascularum,* indicating that this red ooze, was due not to red bacteria but to a red reaction on the part of host-plant. Perhaps the formation of this pigment would not be as noticeable in all varieties of sugar cane as in common green cane. It is not a sign peculiar to this disease of an entirely different nature."

Smith also found yellow and red bundles in canes which he had inoculated and says: "I am inclined to think that the reddening of the bundles is a later stage of the disease than the yellowing".

Smith also found the white bacterial ooze in canes which were received from Australia and says: "No attempt was made to get cultures from the most badly decayed of these canes, because in places the bacterial ooze was nearly white, indicating an extensive mixture of organisms in the stem".

Smith also found the black discolorations in canes which he had inoculated and says: "There are at least 100 bundles affected in the inoculated internode. About half of these are black and a few red, but there are a large number which show the yellow bacterial ooze".

R. Greig Smith made a study of red vascular bundless in 1904 and found a pycnidia bearing fungus and a bacillus which he describe as *B. pseudarabinus.* He says:

"In glucose-gelatin the mould produced a brilliant crimson-scarlet color, and it undoubtedly was the agent which was primarily responsible for the color of the strings. But from the presence of gum in the vessels I was of the opinion that the phenomenon of red gum was brought about by the simultaneous growth of two organisms, a mould and a bacterium. This view was confirmed during the research. It may, however, be mentioned here that every portion of red vascular bundle that was taken did not contain the mould, but did contain slime-forming bacteria; and from this we must conclude that the mould does not accompany the gum along the whole length of the string, but colors the gum which is carried along the vessels, perhaps by sap-pressure, perhaps by bacterial

growth, or that the rapid growth of the bacteria starves out the mould after the color has been produced. At any rate two things are certain: (1) The mould can, under certain conditions, produce the color and cannot produce the slime, and (2) the bacteria do produce the slime''.

He did not reproduce the disease by inoculation but obtained a red color by inoculating a plate with both the fungus and the *B. pseudarabinus*. He did not obtain the color when he inoculated the plate with the fungus and the *B. vascularum*.

The writer does not consider the discoloration of the tissues of importance. These discolorations may result from any condition which interferes with the growth of the cane, insect-injury, disease or as a result of over-maturity, and are frequently found in growing canes which are apparently healthy and normal. The writer has made a large number of cultures from discolored fibro-vascular bundles and other tis ues and while fungi and bacteria are frequently found, many of them do not give a growth of any kind. Furthermore, the fibro-vascular bundles and tissues of infected canes may not show any discoloration or only slight tinting with yellow.

INOCULATIONS

Numerous inoculations from pure culture were made. In general the susceptible canes contracted the disease while the resistant varieties remained healthy. However, in a number of cases the susceptible varieties failed to develop the disease.

Carefully selected infected seed of all the susceptible varieties used in our seed plots were planted in the green house and observed for a period of twelve months. The most severely infected seed pieces died. Some few stools showed a gumming in the young canes but no gumming in the mature canes. Many of the slightly infected seed pieces produced cane without symptoms and were apparently healthy in every way. The medium infected seed pieces produced the disease and gave the following symptoms:

1. An occasional plant showed distortion in coming through the ground.

2. Several plants showed white blotches or white stripes on the leaves.

3. After the formation of the nodes, several varieties showed the leaf streaks (Figs. 8–10) described on page 158.

4. The canes with the leaf streaks showed the gum.

THE PATHOLOGIC HISTOLOGY OF THE HOST

The response of the host to the parasite varies greatly with the variety of the cane and with the age of the tissues at time of infection. Susceptible varieties and young canes or the young tops of older canes show a greater amount of internal injury than resistant varieties and old canes. Smith (**21**) gives a number of excellent illustrations of the morbid anatomy of the host plant but his discussion is very limited.

The specific name of the organism (*vascularum*) indicates that it lives in the vascular tissues and this is true in the case of resistant varieties and in old canes, but it frequently invades the parenchyma tissues of susceptible varieties and of young canes.

In resistant varieties and in old canes, especially the basal part, the organism is usually confined to the tracheary tubes and there is very little or no disintegration of the tissues (Figs. 11, 17). In such cases the injury is physiologic since the plugging of the tubes interferes with the movements of the water in the plants. In more severe cases the cell walls are dissolved, the first to disappear being the cross walls or partitions in the tracheary tubes. In more severe cases the rings and other thickenings inside the tubes are destroyed and fragments are frequently seen in the mass of gum and bacteria which fills the tubes (Figs. 12, 13, 18, 19, 20, 24, 25, 26). In extreme cases the side walls of the tubes are dissolved (Figs. 19, 27) and the bacteria invade the parenchyma tissues (Figs. 14, 16, 21, 22, 23, 29, 30) in which the walls are more or less completely destroyed. The writer has never found the extreme destruction of tissues, except in the very susceptible varieties, such as Otaheite, Cristalina and Rayada, and in young canes and in the tops of old canes. In these very severe cases the young canes and the tops of old canes are killed. The bacteria may be found in any cells of the fibro-vascular bundles except in the sclerenchyma cells (Fig. 17) but there is very seldom any tendency to the disintegration of the tissues, except in the very susceptible varieties and the very young tissues. In the severe cases when the tops gum and die, the tissues in this region are almost entirely disintegrated and form a soft, gumming, slimy mass. The bacteria can also be found in the intercellular spaces of the parenchyma and in parenchyma cells where the cell walls are uninjured. This is especially true in the chlorotic areas of the leaves of infected canes and in the young tissues of susceptible varieties.

The organism appears to travel through the tracheary tubes with

the growth of the canes. It is always most active in the younger parts of the tubes. In fact, it appears to die out in the older parts, leaving the gum which hardens and contracts. The contractions result in a pulling away from the walls of the tube or formation of cracks in the gum (Figs. 24, 26).

The organism follows the fibro-vascular bundles into the leaves and causes the yellowish or whitish stripes along the veins, which have been referred to in the discussion of the symptoms. In severe cases, it causes a disintegration of the cell walls and spreads into the surrounding tissues as in the cane (Fig. 27). When the leaves are fully spread and exposed to the wind, the dead tissues become dry (Figs. 9 & 10). When the leaves become severely infected before they unroll, a slimy mass of bacteria and a disintegrating tissues is the result (Fig. 22). In young canes the entire shoot dies, while in old canes the top dies and gives the characteristic dead top symptom.

A comparative study of sections of a large number of varieties leads the writer to believe that the more fibrous canes are more resistant than the canes with a low fibrous content. The old varieties of canes were of low fibre content and were well adapted to old machinery for grinding. The improved machinery enables us to grind the high fiber canes and thus to eliminate the old varieties which are so susceptible to gummosis, mosaic and other diseases.

FIELD PLOT TESTS

Through the kindness of the authorities of Central Columbia near Maunabo, a plot of one and one-half acres of good uniform cane land was set aside for experimental tests. This was planted to varieties in rows of forty-five stools, except in the case of a few varieties of which there was a shortage of seed cuttings. In these cases we used one-half or one-third rows. Two seed pieces were planted in each stool. Every third row was planted with infected Crystalina cuttings so that each variety came in contact with infected cane on one side. The plantings were made October 16th, 1925, and observations to determine symptoms were made from time to time during the growing season. The first cutting was made February 16th, 1927, and the second February 7th, 1928.

The symptoms during the growing season showed little evidence of the disease and did not correspond with the percentages of gumming at time of cutting. Therefore, the writer is of the opinion that external symptoms during the growing season are a very poor index of the amount of infection. In every case that has come to the

attention of the writer, the percentage of gummosis in susceptible varieties was higher than indicated by external symptoms.

Considerable difficulty was experienced in determining a method for estimating percentages of infection: (1) because a considerable number of stools died and it was impossible to determine the cause with any degree of certainty; and (2) because many young canes died from this and possibly other causes and cannot be included in the count; and (3) because the infections were very slight in some cases and very severe in others. *It was finally decided to base the percentages on the actual number of living canes showing gum as compared with the number which did not show gum at time of cutting.*

All of these facts are shown in Table 1. The mill records are of little importance because it is well known that these figures will vary with the ages of the canes at time of cutting.

NOTE: After this paper was in type some question was raised as to the desirability of giving the number of stools infected at time of first cutting. Those who are interested in this data will find the figures in Table VI (page 179). However, it should be remembered that the infections in some stools may occur in only one cane while in other stools the infections may occur in all the canes. Therefore, the writer is of the opinion that the percentages given in Table I are of greater value than the data in Table VI because it gives a better idea of relative resistance and susceptibility.

TABLE I

	1926	1927		1928		1927		
						Mill record		
	Number of stools planted	Number of stools living	Percentage of disease	Number of stools living	Percentage of disease	Brix	Sucr.	Pur.
PR-491	45	0		—				
H-109	15	15	100	11	63.7	15.7	13.78	87.7
PR-450	45	45	100	33	42.4	15 2	13 17	86 6
PR-260	45	45	100	36	22 2	13.7	9.14	66.7
B-6292	75	75	100	67	10 3	18 2	15 75	86.6
Otaheiti	45	6	100	2	100	13 8	10.61	76 0
B-6808	45	40	86	37	0	18.6	15.02	80.7
Cristalina	1260	1130	85	942	9.3			
PR-487	15	13	80	12	91.6	17.5	13.06	74.6
D-504	15	12	66	8	12.5	16 8	13 90	85.0
B-7032	45	31	66	31	0	16 3	13 90	85 2
EK-28	15	14	63	14	50	20	17 05	85.1
D-1135	45	39	64	39	0	16 7	13 18	78 9
Rayada	90	70	55	64	3 1	16 6	14 20	85.0
St. Kitts	15	14	45	14	0	19 8	19 00	95 9
D-109	90	77	42	77	0	17.5	14 58	83 3
PR-292	15	9	33	8	0	16.6	14 20	85.5
X-64	45	35	33	26	7.7	15 8	12 75	80 6
Ba-11569	45	4	28.5	42	7.1	20.9	19.64	93.9
Badila	45	41	27	41	0	15 6	12.37	79.2
B-3405	45	40	25	40	12.5	17.9	12.42	69.4
B-3696	45	40	17.5	38	0	17.7	13.61	76 8
B-3412	45	40	17.5	40	0	18 6	13.86	74.5
PR-492	90	66	17	66	0	18.0	14 79	82 1
PR-328	45	26	15	25	4	19.7	17 28	87.7
PR-417	45	38	13.1	34	0	20 6	17.52	85.2
GC-493	15	8	13	7	0	19.4	14 72	75.9
B-1809	45	41	12.2	38	0	19 6	17.23	87.9
Yel. Cal	45	35	14.3	35	0	19.4	13 52	69.6
B-1753	15	12	8	8	0	18.1	16 27	87 8
PR-383	45	27	7 4	25	0	19.5	15 94	81.7
D-117	45	30	6 6	30	0	17 8	15 82	88 8
D-433	45	33	6 1	33	0	17 7	12 00	79 2
FC-214	45	33	6	33	0	18 2	15 90	87 3
PR-219	20	17	5.9	17	6	18 9	16 45	87 0
PR-329	45	34	5.9	31	0	14 7	9.67	75 7
FC-306	75	54	9.5	54	0	17.4	15 50	89.5
SC-12 4	90	77	2	76	0	21 7	19 77	91 4
P R. 202	15	12	8 3	12	0	19.4	16 91	81 7
PR-729	45	32	1	30	0	16.7	13 24	79 2
PR-358	45	38	1	37	0	16 9	15 44	91.3
PR-230	15	11	1	9	0	18 2	14 79	81.2
PR-67	45	45	1	40	0	18 4	14 79	81 2
B-208	45	42	1	34	0	19 4	16 91	87.1
BH-10 12	160	133	1	130	0	21.9	20 37	93.0
PR-318	15	12	0	12	0			
D-448	45	41	0	35	0			
POJ-979	15	7	0	7	0	17.7	15 30	86.40
POJ-826	15	9	0	8	0	18 8	17 20	91.4
POJ-234	15	13	0	13	0	17 8	14.87	87 4
POJ-228	15	14	0	13	0	16 0	13 93	87 0
M-36	22	22	0	22	0	18 6	16 04	86.2
Uba	45		0	45	45	19.9	15 22	76.4

A comparison of the amount of infection at first and second cutting is shown a little more clearly in Table II which gives percentages only.

TABLE II

First Cutting.	Second Cutting.
I. 100 per cent infection—	
PR–260	
PR–460	PR–491
PR–491	PR–487
H–109	
B–6292	
Otaheiti or Blanca	Otaheiti or Blanca
II. 80 to 90 per cent infection—	
PR–487	
B–6308	
Cristalina	
III. 60 to 70 per cent infection—	
PR–504	
Ba–6032	
D–1135	H–109
EK–28	
IV. 50 to 60 per cent infection—	
	EK–(exact 50%)
Rayada	Cristalina
V. 40 to 50 per cent infection—	
D–109	PR–460
St. Kitts	
VI. 30 to 40 per cent infection—	
PR–292	Cristalina
X–62	(Some rows. Many other rows not infected.)
VII. 20 to 30 per cent infection—	
B–3405	PR–260
Ba–11569	Cristalina (Some rows.)
Badila	
VIII. 10 to 20 per cent infection—	
PR–492	Cristalina (Some rows.)
PR–417	D–504
PR–328	B–3405
B–3696	B–6292
B–3412	
B–1809	
Yellow Caledonia	
GC–493	

TABLE II—Continued

First Cutting.	Second Cutting.
IX. 1 to 10 per cent infection—	
PR–329	X–62
PR–333	Cristalina (9 rows)
PR–219	PR–328
FC–306	PR–219
FC–214	Ba–11569
D–433	Rayada
D–117	
B–1753	
SC–12(4)	
PR–202	
X. Less than 1 per cent infection—	
PR–729	
PR–358	
PR–202	
PR–230	
PR–67	
B–208	
BH–10(12)	
XI. No infection—	
PR–318	POJ–979
D–448	POJ–234
POJ–979	PR–492
POJ–826	Crist. (10)*
POJ–234	PR–729
POJ–228	D–433
M–36	B–6032
Uba	FC–214
	St. Kitts
	B–3696
	B–67
	B–3412
	PR–333
	BH–10(12)(4)*
	D–109
	D–117
	SC–12(4)*
	Yellow Cal.
	PR–230
	PR–292
	POJ–234
	POJ–826
	GC–493
	FC–306(2)*
	PR–318
	SC–12(4)(2)*
	PR–729
	B–6308
	B–1753
	B–208
	B–1809
	PR–492
	PR–417
	PR–329
	PR–358
	D–1135
	D–448
	Badila
	Uba
	PR–202
	M–36

* The figure in parenthesis indicated number of rows without infection.

It will be readily seen that the amount of infection was much less at time of second cutting that at time of first cutting. That only one variety (P. R. 487) showed a higher percentage of infection at

second cutting and only one variety (P. R. 219) showed the same percentage at second cutting. All the other varieties showed a lower percentage at second cutting except P. R. 491 in which all plant were killed. The tendency for the disease to be more severe on plant than on ratoon cane and to vary in amount from year to year has been noted by others in Porto Rico with whom the writer has talked. This has also been noted by workers in other parts of the world. We have already called attention to the fact that the reports indicate a reduction of the disease in Porto Rico during the year following 1921, until the severe outbreak in 1925. In one of Cobbs early Australia publications he says: "All symptoms of gumming seem to be more pronounced when they occur in plant cane than when they occur in ratoons." In a more recent publication North of Australia says that the disease apparently disappears in some places and reappears in others.

All of PR-491 died the first year, presumably as a result of this disease.

Only six stools of Otaheiti were living at the end of the first year and only two at the end of the second year and there was a 100 per cent infection in both instances. The second Brazil report (1892) says: "The unfortunate 'Otaheiti' cane, or Cayanna' cane, as it is called in Brazil, was here as elsewhere the principal victim, and nine-tenths of the cane grown was of this kind". Matz also records it as strongly susceptible.

H–109, PR–460, PR–260 and B–6292 all showed a 100 per cent infection the first year but with no loss of stools. However, the number of living stools and also the percentage of infection were reduced the second year. The writer is unable to give any explanation as to the cause of the death of these stools, unless the weaker plants died as a result of the disease the first year. The fact that the cane is more severe on plant than on ratoon cane, emphasizes the importance of using disease free cane for planting.

It will be noted that of the 53 varieties used (not including Cristalina), that there was a reduction of living stools during the second year. The tonnage was also less the second year. It is the opinion of the writer that a considerable part of this loss was due to the disease and that a very small part was due to other causes. This also emphasizes the importance of making every possible effort to control the disease.

It will also be noted that all varieties except Otaheiti and PR-

219, showed a lower infection in the second than the first year. Was this due to a killing of the weaker individuals during the first year or to other agencies? We do not have data to answer this question which must be left open for future study.

The notes of the writer record the first year infections of one row of BH–10 (12), Ba-11569, B–6308, PR–202 and one row of Cristalina as slight. The infections on the individuals canes were slight but the percentage of infected canes was as indicated in table III.

TABLE III

Variety	First cutting	Second cutting
Cristalina *	100	61
B-6308	86	0
Ba-11569	28. 5	7.1
PR-202	8.3	0
BH-10 (12)	5.9	0

* One row only, 45 stools.

Therefore it will be readily seen that relative severity of infection and percentage are not the same. Two varieties or two rows of the same variety may show the same percentage of infection but the infection may be more severe in one case than in the other. These varieties showed a high percentage of infected canes but the infections were very slight.

At the time of first cutting only one variety showed chlorosis. GC–493 showed a 13 per cent infection and one stool with chlorosis.

The writer used twenty-three varieties that had been used by Matz and the comparative results may be of interest. However, it should be noted that the methods were different. Matz inoculated the canes with pure cultures of the organism while the writer planted apparently healthy seed cuttings in rows parallel and five feet distant from rows planted with infected seed cuttings of Cristalina. The writer has no data by which it is possible to judge the relative merits of the two systems but Matz says in regard to his own system:

"This method is not quite satisfactory since it does not represent a truly natural state of affairs, but it shows the possible susceptibility of each variety" The comparative results on the twenty-three varieties is shown in Table IV. The grouping in the first column is according to Matz; the Roman numerals in the second and third columns correspond to the Roman numerals used in Table II.

TABLE IV

Matz	Cook	
	First year	Second year
I. Strongly susceptible		
Otaheti	I	I
Rayada	IV	IX
Cristalina	II	*
PR-491	I	I
II. Slightly susceptible		
PR-260	I	VII
B-3405	VII	VIII
PR-328	VIII	IX
III. Susceptible when young.		
B-208	X	XI
IV. Resistant or immune.		
Uba	XI	XI
D-448	XI	XI
Yellow Caledonia	VIII	XI
B-3412	VIII	XI
D-117	IX	XI
B-6292	I	VIII
D-109	V	XI
PR-202	IX	XI
PR-230	X	XI
BH-10(12)	X	XI
PR-333	IX	XI
PR-292	VI	XI
PR-318	XI	XI
PR-417	VIII	XI
PR-219	IX	IX

* Cristalina gave variable percentage at second cutting.

The results are not radically different. The most striking differences were with two varieties:

TABLE V

Variety	Matz	Cook	
		First Year	Second Years
		Percent	Percent
PR-260	Slightly susceptible	100	22.2
B-6292	Resistant or immune	100	10.3

Although Matz did not do row-to-row planting with diseased and healthy seed cuttings he did conduct one test comparable to the writer's work. This is best described in his own words:

"Stools of cane which contained several diseased stalks were dug up and all the stalks and shoots cut back. The stubble with their roots were transplanted amongst young healthy canes in an isolated field. It was noted that some young shoots which came up from

those diseased stools showed at the very beginning the symptoms of gum disease, and what is more significant the disease was later found in the adjacent healthy stools as well. It is therefore evident that the infected ratoons left in the field constitutes a positive source of infection and that the disease can be carried over from these to the young canes of healthy stools. The possibilities are, therefore, that the disease can be transferred to growing cane by insects, by the cutting instruments and by driving rains, but by eliminating the diseased ratoons the primary source of infection is destroyed, since the soil does not form a favorable abode for the bacterium. Artificial inoculations in the roots of susceptible canes gave negative results. This can not be explained on the ground that the acidity of the soil does not favor growth in the bacterium''.

The writer made plantings of diseased seed cuttings of all the varieties recorded in Table I in the green house at Río Piedras and found that in most cases, slightly infected seed cuttings gave apparently healthy plants, that the majority of severely infected seed cuttings died and that the medium infected seed cuttings gave the greatest number of diseased plants. Percentage records were not made in this test because the number of cuttings of each variety was too small for satisfactory percentages.

The writer also inoculated growing plants and seed cuttings with pure cultures of the organism but found the results very irregular. In many cases susceptible varieties did not always produce diseased plants.

No experimental work on transmission was conducted by the writer. However, it is very evident that the disease can be transmitted by the seed cuttings. The character of the organism and nature of the disease are such that we would expect it to be carried on the cutting knives and by insects but we have no data on either point in Porto Rico. However, the work of both Matz and the writer show very clearly that the disease can be transmitted from stool to stool and row to row. The writer planted severely infected seed pieces in large pots. Some of them died without producing shoots. Two months later, a clear gum was found on freshly cut surfaces of these seed pieces. Cultures were made and the organism found to be very much attenuated but capable of transmitting the disease. No effort was made to determine whether the organism could be transmitted in the soil but a test by Matz should be mentioned in this connection. He says:

''Gum-diseased cane pieces, the buds of which were removed split

and these were tied to healthy seed planted in new soil. Twenty seed of each of the varieties Caña Colorada, Yellow Caledonia, Rayada, Otaheiti and PR–260 were used in the trial, each variety being planted in a separate row. In addition five seeds of each of the above varieties were planted in the same rows but alone without infected cane. Practically all the healthy seed in this whole planting germinated and no sign of disease was noticeable in the young plants in spite of the fact that at first their buds and later their roots had been in contact with gum diseased cane pieces which were gradually decaying in the soil. They all made a good normal growth, and when the whole plot was harvested at the age of eight months, there were no traces of gumming in the stalks of any of the varieties used. The ratoons of these canes sprouted normally and no disease symptoms were noticeable in them when they were cut 10 months later. Apparently the soil is not the proper means through which infection might be carried to the roots of healthy seed. In another experiment diseased seeds were planted with the view to allow those to sprout, as only 25 per cent germinated; healthy Otaheiti seeds were planted in the holes of the ungerminated seed. There was no gum disease produced in the replants of the susceptible Otaheite in this manner. However, when *Bacterium vascularum*, isolated from diseased cane, was introduced with a needle into the young leaf spindle of Otaheite and Rayada canes growing in the field the disease was reproduced with all its symptoms. That indicates that the air route is the path of transmission for this disease."

DISCUSSION

Our studies on this disease show that it is very serious and very destructive on susceptible varieties. Although it may vary somewhat in severity from year to year, it causes a reduced yield in tonnage and interferes with the crystallization of the sugar in the mills. Although the amount of the disease may be greatly reduced and it may even disappear at times, the planting of infected seed is likely to result in losses in the succeeding crop. In all cases that have come under our observation, the percentage of infection was higher in plant cane than in ratoons but the tonnage was less in the ratoon than in the plant canes.

There is a very great variation in the susceptibility of varieties as shown in the tables of this paper. It will be noted that some of our old varieties, such as Otaheiti, Cristalina and Rayada are very susceptible. These varieties appear to be low in fiber in comparison

with the very resistant canes. Possibly their low fiber content is the cause of their being such great favorites with the early growers who were compelled to use the crude and inefficient mills of the past. Improved machinery makes it possible to grind the more fibrous canes which have been coming in use in recent years.

An examination of the tables shows that some of our favorite canes, such as BH–10 (12) and SC–12 (4) are very resistant to this disease. Therefore, it appears that we can control the disease by growing resistant or immune varieties which are as good or better than the susceptible varieties.

We recommend (1) that our growers abandon varieties that show a susceptibility of more than 5 per cent; (2) that they do not use seed from crops in which the disease has been found; and (3) that diseased crops be destroyed and the fields replanted with healthy seed of resistant or immune varieties.

The presence of this disease on the island will prevent the introduction or development of new, improved varieties which are susceptible to the disease. The introduction of an insect capable of carrying the disease might result in serious complications. Therefore, our growers should use every possible measure for the eradication of the disease.

SUMMARY

1. Gummosis of sugar cane is a bacterial disease, caused by *Bacterium vascularum* (Cobb) Grieg Smith.

2. The organism lives in the tracheary tissues of the fibro-vascular bundles. Sometimes it dissolves the cell walls and spreads into the surrounding tissues of susceptible canes and into young tissues of somewhat resistant canes.

3. The organism produces a gum which oozes out of the cut ends of the infected canes.

4. The disease kills many young canes and causes a reduced yield, varying with the susceptibility of the variety and the severity of the attack.

5. The gum also interferes with the crystallization of the sugar in the mills.

6. The percentage of infection is higher on plant than on ratoon cane but in all cases that have come under our observation, the yield on the ratoons was reduced.

7. The leaf symptoms are more pronounced during wet than during dry weather.

8. Many of our best varieties in Porto Rico are immune or highly resistant. Therefore, the disease is not a serious problem if these varieties are used.

9. Immune or resistant varieties should be used in Porto Rico. Infected cane should never be used for commercial planting.

The writer wishes to express his thanks to Commissioner C. E. Chardon for valuable advice and assistance, to the proprietors and manager of Central Columbia for land and cooperation in the field experiments and to Mr. Francisco Ortiz who had charge of the cultivation of the field for the very valuable asistance rendered by him.

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EXPLANATION OF PLATES

PLATE I

Fig. 1. Cane showing the exudation of the gum.
Figs. 2–6. Chlorosis due to gummosis on young cane of 15 inches or less.

PLATE II

Figs. 7–10. Leaf symptoms on older cane.

PLATE III

Fig. 11. Section at base of cane showing the organism confined to the tracheary tubes.
Fig. 12. Section of fibro-vascular bundle showing the beginning of the disintegration of the cell-walls.

Fig. 13. Longitudinal section showing disintegration of the cell-walls.
Fig. 14. Section of parenchyma tissue showing cells filled with the organism and slight disintegration of cell-walls.
Fig. 15. Section through a dying top showing the disintegration of the host tissues into a slimy mass containing fragments of cell-walls, gum and bacteria.
Fig. 16. Longitudinal section through parenchyma tissue showing disintegration of cell-walls.

PLATE IV

Fig. 17. Cross section of part of fibro-vascular bundle showing distribution of the bacteria in the cells.
Figs. 18–19. Longitudinal section of fibro-vascular bundle showing disintegration of cell-walls and tracheary rings.
Fig. 20. Cross section of fibro-vascular bundle in the streak of a leaf in which the bundle is almost entirely disintegrated.
Fig. 21. Parenchyma cell showing bacteria.

PLATE V

Figs. 22–23. Parenchyma tissue undergoing disintegration.
Fig. 24. Longitudinal section in which the old gum has hardened and pulled away from the wall or one side of the tracheary tube. No bacteria visible in this section.
Fig. 25. Cross section showing same as in figure 31.
Fig. 26. Cross section showing same as in figure 32 except that the hardened gum has cracked.

TABLE VI

	Number of living stools	Number of stools showing infection
H–109	15	15
PR–460	45	45
PR–260	45	45
B–6292	75	75
Otaheiti	6	6
B–6308	40	39
PR–487	13	12
D–504	12	10
B–6032	31	3
EK–28	14	14
D–1135	39	29
Rayada	70	49
St. Kitts	14	10
D–109	77	38
PR–292	9	5
X–62	35	30
Ba–11569	42	12
Badila	41	11
B–3405	40	10
B–3696	40	7
B–3412	40	7
PR–492	66	12
PR–328	26	7
PR–417	38	5
GC–493	8	2
B–1809	41	5
Yel. Cal.	35	5
B–1753	12	4
PR–333	27	2
D–117	30	2
D–433	33	6
FC–214	33	3
PR–219	17	1
PR–329	34	2
FC–306	54	7
SC–12(4)	77	2
PR–202	12	12

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PLATE I

PLATE II

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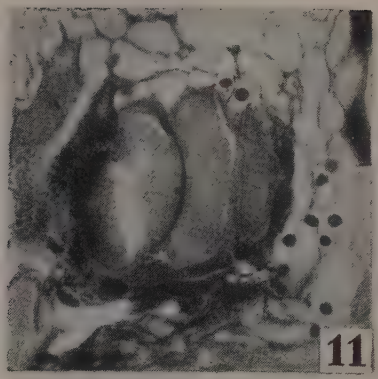
Illustrations by Mario BRAU.

PLATE III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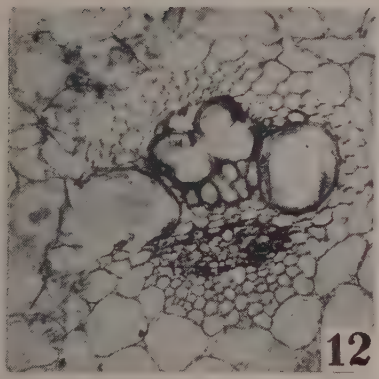
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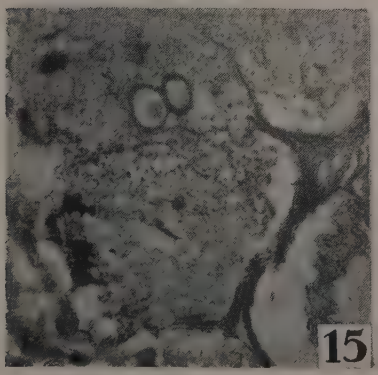
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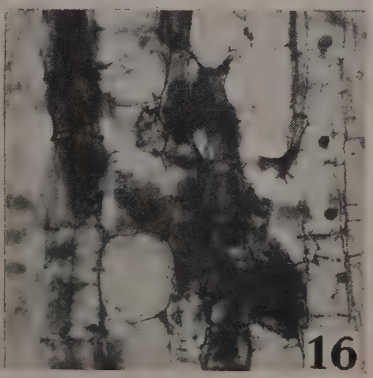
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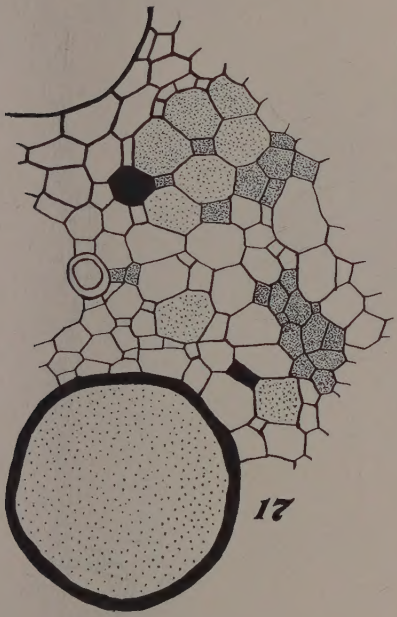
PLATE IV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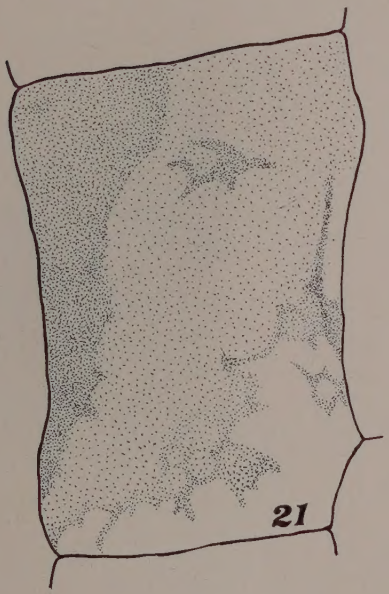
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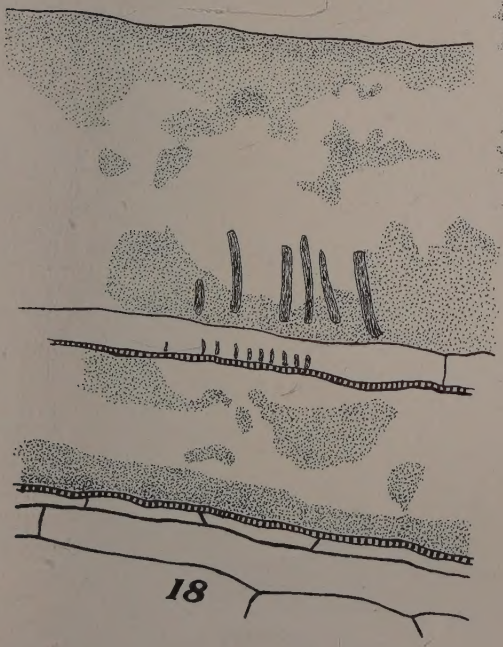
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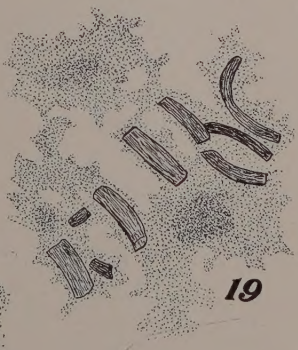
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PLATE V

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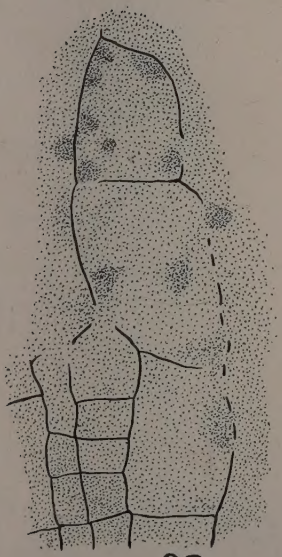

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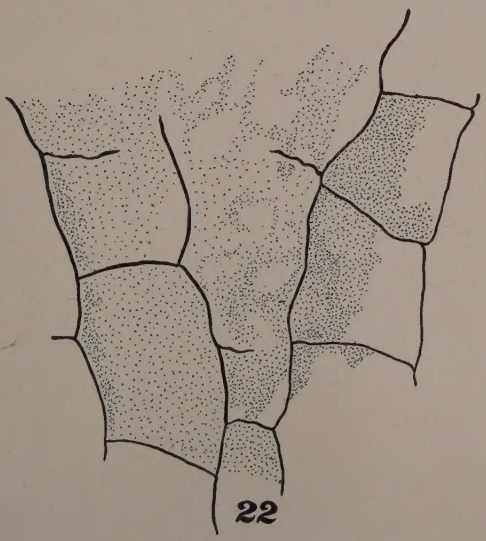

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